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THE HAMILTON STUDY: EFFECT OF PARTICLE SIZE ON RESPIRATORY HEALTH IN CHILDREN*

L. D. PENGELLY[1], C. H. GOLDSMITH[2], A. T. KERIGAN[1],
W. FURLONG[1] AND S. A. TOPLACK[1]

DEPARTMENTS OF MEDICINE[1], and CLINICAL EPIDEMIOLOGY AND BIOSTATISTICS[2]

McMASTER UNIVERSITY,
HAMILTON, ONTARIO, CANADA

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ADDRESS FOR CORRESPONDENCE:

Dr. L. D. Pengelly
Urban Air Environment Group
Room 3E27
McMaster University
Health Sciences Centre
Hamilton, Ontario
L8N 3Z5

*SUPPORTED BY: Province of Ontario: Ministry of the Environment
Ministry of Health
Canada : Health and Welfare

ABSTRACT

Hamilton, Ontario, is an industrial city with a population of 300,000 situated at the western end of Lake Ontario. The level of particle pollution has historically been high, a portion of which has been attributed to the activities of Canada's two largest iron and steel mills located here. Although in the last two decades air quality has improved markedly, there has been a continuing community concern about the effects of air pollution on health. From 1979 to 1982, we carried out an epidemiological study of the respiratory health of a cohort of over 3500 elementary school children in whom we also determined the extent to which they were exposed to ambient particles of different sizes. Respiratory health was assessed by determining each child's respiratory symptoms and health history using a questionnaire administered to the parents. In addition, comprehensive tests of pulmonary function were carried out on the children at their schools. Particle size and concentration were measured using two networks: one of 7 to 9 Andersen 2000 cascade impactors, and another of 27 GMW Hi-Vol TSP samplers. Both networks were distributed over the area of the city. Other environmental factors thought to "confound" the relationship between respiratory health and air pollution were also examined via information collected in the parental questionnaire; e.g., parental

smoking, use of gas for cooking, socio-economic status. Results of a cross-sectional analysis of the data from the cohort in the second year of the study showed a strong effect of maternal smoking on the prevalence of cough and bronchitis, with little air pollution effect on these variables. Two pulmonary function variables, peak flow (adjusted for height) and maximum expiratory flow at 75% VC (adjusted for height) showed an association with the presence of fine particles.

BACKGROUND

Hamilton, Ontario, is an industrial city of 300,000 people located 80 kilometers to the west of Toronto, at the western end of Lake Ontario. The largest two steel mills in Canada are located here, and the city has had an historical problem of particulate pollution, first documented in the mid 1950's (1). Early studies from our laboratory showed that the persistent aerosol in the atmosphere over Hamilton could be visualized and its extent and altitude measured by a simple technique using a searchlight (2). In this study we demonstrated that aerosols were frequently trapped in inversion layers over the city, especially when the wind came from the northeast over the cold lake. We were able to demonstrate distinct layering of the aerosol from ground level up to several hundred meters on many occasions during a study period in the summer.

We also operated a pollution sampling network using Hi-Vol samplers for "Total Suspended Particulate" (TSP) and Andersen cascade impactors to measure particle size distribution (PSD). The station which was the farthest south in the network was on the tip of Long Point and was located virtually in the centre of Lake Erie. By operating

the network simultaneously on a 6-day cycle we were able to show that both the concentration and size of the particles moving from Lake Erie in the direction of the prevailing wind toward Hamilton increased substantially. The greatest increase began at the city limits, and substantial gradients were observed while moving in a northeasterly direction over the industrial area (3).

In 1977 a pilot study was carried out in preparation for the present study, in which we made measurements of sulphur dioxide (SO2) as well as TSP at an additional location within the city, which confirmed our impression of a substantial gradient in both particulate and SO2 pollution from the southwest to the northeast (4). Thus before we started the present study, we had good evidence that there were substantial gradients of TSP, PSD and SO2, the highest levels being more than twice the lowest levels. As a consequence, we decided to make use of these gradients to carry out an epidemiological study of the effects of air pollution on respiratory health.

METHOD

We decided to carry out our study by examining the effects of air pollution and other environmental factors on the

respiratory health of a sample of elementary school children in the city of Hamilton. We were aware that many factors other than air pollution influenced respiratory health, so our study was set up to examine the relative contribution of many environmental factors on the respiratory health of our school children.

Sample

There are many reasons for the use of young children. It is thought that the growing lung may be more sensitive to insult than the adult lung, and therefore that the child population is more at risk from adverse air quality. In addition, with children, there is likely to be a much better characterization of the environmental conditions in which the children have grown, either before or during the period of study. Children are less exposed directly than adults to occupational or recreational sources of toxic agents, and the prevalence of self-smoking in Canadian children (excluding Native children) under the age of 11 is less than 3% (5).

Accordingly, we chose to study children enrolled in schools of the Board of Education of the City of Hamilton in grades 2, 3 and 4 who had not reached their 11th birthday by

December 31, 1978. There were over 8400 children in these grades, and sample size calculations indicated that we would require 3200 in a stratified random sample (10). An attempt was made to have the children uniformly distributed over 4 "quadrants" of the city (west upper - WU, east upper - EU, west lower - WL, and east lower - EL), as shown in Figure 1. Figure 1A shows the areas of the city, and the locations of the aerosol samplers. Figure 1B shows the distribution of the schools with respect to the samplers. In the second year (Year II) of the study (reported here), which took place in 1980, we added children of the same age as the original cohort from three schools in the industrial core (IC) of the city, a subsection of the EL quadrant. The total number of children studied in Year II was 3588. Parents were interviewed and children studied once each year for a total of three times, essentially in the calendar years 1979, 1980, and 1981. Further details of the sample are given in a previous report (10).

Measurements of Suspended Particles

For the purposes of the epidemiological study, we set up a pollution sampling network to measure TSP at 27 sites and PSD at 7-9 sites. At the time we began the study, the Ontario Ministry of the Environment (OME) operated 11 TSP sites in an abatement-oriented network in the industrial and commercial parts of the city, and had no ongoing measurements of particle size distribution. Since for our purposes we had need of an estimate of an exposure which was more population based, we found it necessary to complement the OME network with 16 TSP sites of our own, and to set up a network of PSD samplers (7 in 1979 and 1980, 9 in 1981). Our TSP sites were for the most part located in residential areas, but the PSD locations were distributed across the city.

TSP was measured with standard Hi-Vol samplers (General Metal Works GMWL-2000H) using glass fibre filter paper, and PSD was measured using the same housing, but with a 4-stage cascade impactor designed to operate at 20 cfm (Andersen 2000) (6). Flow recorders were used on all TSP samplers. PSD flow was set to 20 cfm before sampling by means of variable auto transformers on all PSD samplers, and was measured and recorded after sampling. Filters were

conditioned at known humidity before and after exposure, and weighed with a Mettler Gram-matic balance. Both networks were operated every six days synchronously with the North American Synoptic Six-day Cycle. Quality control procedures were used, which will be reported elsewhere.

Summarization of Data

For TSP, the results for each site were expressed in ug/m^3, and this was recorded together with the site identification number and the date of sampling. The five or six values for a given site were used to generate a monthly geometric mean value for that site. If fewer than three values were available for a site for a given month, the data for that month were declared "missing". This criterion also held for PSD. In order to summarize the results of the measurements obtained by the PSD network, we chose to obtain monthly average values in the following way. Since the sampler collected aerosol on four separate impactor stages as well as on a backup filter, on a given sampling day we could measure it in each of five size ranges, and this was expressed in micrograms per cubic meter. To obtain a summary of information for a month, the loadings on corresponding filters were added together for the five or six sample days, and a mean monthly loading for each

stage or filter was calculated. From the five monthly loadings, aerodynamic mass median diameter could be calculated in the standard manner and expressed as MMD in micrometers (um) (6). In addition, we decided to add the loadings of the first two stages together including particles greater than 3.3 um, and referred to this as the "coarse fraction" (CF). We added the loadings of the third and fourth stage as well as the backup filter together and referred to this as the fine fraction (FF) (i.e. that less than 3.3 um). Thus for a given month, we had a measure of MMD obtained from all the loadings for that month, and a measure of coarse and fine fraction which was obtained from the sum of the corresponding loadings for that month, and expressed as ug/m^3.

Comparison of PSD with Dichotomous Sampler

Our measurements of particle size and distribution depend on the characteristics of the Andersen 2000 cascade impactor. We understand that particle separation may be underestimated by virtue of particle "bounce", especially from the first to the second stage. By combining these two stage loadings into the "coarse fraction" we believe we have minimized this effect by using the CF/FF method of separating loadings. In addition, in 1983-84 we had the

opportunity to compare the measurements of a Sierra Series 245 dichotomous sampler operated by the OME with those from our Andersen 2000 at the same location. The comparison took place over a full year, and there were 41 complete measurements of both instruments available in this period. The results of this comparison showed that the concentration of the fine fraction of the dichotomous sampler was highly significantly correlated with the Andersen fine fraction. A plot of these data is shown in Figure 2. The regression equation corresponding to these data is:

$$\text{DICHOT FF} = -7.5 + 0.65\ \text{ANDERSEN FF} \quad (R^2 = 50.1\%).$$

Correspondence was also found between the two measurements of coarse fraction:

$$\text{DICHOT CF} = -4.7 + 0.29\ \text{ANDERSEN CF} \quad (R^2 = 30.3\%).$$

Analysis of Data from Air Pollution Networks

Our objective was to determine an exposure which was as closely as possible unique to each child in the study. For practical purposes we decided to use a time resolution of one month, and a spatial resolution determined by the

location of the school the child attended at the time the child was tested. Thus we had need of a method by which we could estimate a monthly exposure on a school-by-school basis.

Using the monthly values obtained for each site for each variable (TSP, MMD, CF, FF), a response surface was calculated which expressed in three-dimensional terms (vertical direction - aerosol variable; horizontal "x" - easting geographical co-ordinate; horizontal "y" - northing geographical co-ordinate) an estimate of the distribution of the aerosol as a function of the geographical plane of the city. For MMD, CF and FF the form of the response surface model was:

$$MMD = B0 + B1(E) + B2(N) + B12(EXN) + e$$

where MMD is the response value,
B0, B1, B2, and B12 are regression coefficients,
E and N are normalized easting and northing geographical coordinates, and
e is the error term

In addition, the coefficient of determination of the surface could be calculated, indicating the proportion of the variation in the data explained by the model we fitted.

In most cases over 2/3 of the variability in the data was accounted for by the fitted model. Details of this model will be presented in a separate report.

To calculate an exposure value for an individual child, an annual mean was determined by taking the arithmetic mean of monthly values of a given variable for the school location at the time of testing: for the month of testing, the 9 months preceding it, and the 2 months following it. The yearly average was used because we were looking for long-term or chronic effects both in respiratory symptoms and as indicators of these in pulmonary function, and because plots of yearly air quality measurements indicated that seasonal cycles repeated within the years. Although our original design for this study called for a full year of air quality data collection before the collection of health data began, the funding agencies did not permit this. Thus we had to start up the network at the same time as starting health measurements. Thus for the second year of the study (reported here) we did not have a full year of air quality data in advance of the testing date of about 25% of our cohort. However, examination of the seasonal trends of air quality data showed that the spatial patterns repeated from year to year (see Figure 4). Thus to ensure the same exposure calculation for all of the cohort, and to

be able to use the full cohort, we included the two months following testing with the 10 previous ones in the exposure calculation. Our assumption was that although we might not obtain the same absolute exposure value as that from the previous 12 months, nevertheless the spatial differences in exposure the children experienced would be very similar.

Respiratory Health

The respiratory health of the children, and home environmental and socio-economic variables were assessed by means of a questionnaire administered to the children's parents. Pulmonary function tests were conducted in the schools after having received consent from the parents of the children. Interviews and tests were conducted once a year for a period of three years.

Information from the questionnaire included questions on the recent (in the last year) respiratory health and symptoms of the child (cough, wheeze/asthma, lower respiratory infection), earlier history (birth weight, hospitalization as an infant with respiratory disease), family respiratory health (mother and father cough, siblings with respiratory illness), home environment (others' smoking, use of gas stoves, crowding), and

socio-economic variables (household income, mother/father occupational status).

Pulmonary function testing was carried out by two teams of testers with two sets of equipment (Hewlett-Packard 47804A Pulmonary Calculator System) operating simultaneously; one in the lower part of the city and one in the upper part of the city. Pairs of schools were selected at random from the lists of schools to be tested. The exception to this was the addition of the 3 IC schools at the end of Year II. After a pair of schools were studied, the teams crossed over and moved to the opposite section of the city. Three different kinds of pulmonary function tests were carried out: the forced vital capacity; the multiple-breath nitrogen washout; and the single-breath nitrogen washout or so-called "closing volume". A description of the use of these measurements in epidemiological studies has been given by Ferris and colleagues, and by Sterk et al. (7, 8). For the purposes of this report we include the results of the measurements of peak expiratory flow (PF), and the maximum expiratory flow when 75% of the vital capacity has been exhaled (MEF 75). Peak flow is an effort-dependent measure related to large airway function, whereas MEF 75 is influenced both by small airway function and the elastic recoil of the lung (7). A randon 5% of all children had

their pulmonary function retested. The mean difference between the two measurements of PF was less than 3% of the value of the first measurement. The same was true for MEF75.

RESULTS

Suspended Particles

Figure 3 shows contours of mass median diameter obtained using the response surface model for the month of May 1980. Each small number which defines the contour line represents twice the mass median diameter defined by that line; i.e., "8" = 4 um MMD. The particle size to which the children were exposed was calculated on the basis of monthly maps of this type.

For 3 of the 39 months from January 1979 to March 1982 data were available from only 4 sites, and from these data response surfaces could not be calculated. For these months, particle size was calculated from the mean of the values obtained for the 4 sites, and was taken to be uniform across the city. For 36 of the 39 months, data were available from 6 or more sites, and response surfaces could be calculated. In 19 out of 36 months, the spatial

coefficients of the sites were found to be significant at the $p<0.05$ level.

We found that the pattern of the distribution of particle size and fine and coarse fraction was similar from year to year, as is shown in Figures 4 and 5. Figure 4 shows the calendar yearly response surface contours for the three years 1979, 1980 and 1981. Although the nature of the contours are determined in part by the model, it is apparent that the shapes of the contours are similar from year to year. The southwest-northeast axis of symmetry probably relates to the two dominant wind directions: southwest (35% of the time) and northeast (11% of the time) (9). It is clear from these contours that a substantial gradient exists over the city. In Figure 5, the numbers on each contour represent tens of micrograms per cubic meter. For the years 1979 and 1980 the patterns are quite stable, with substantial gradients in both fractions. In 1981 however, when a recession and a prolonged steel strike reduced industrial activity, the reduction in the gradient for the coarse fraction was much more marked than that seen for the fine fraction.

Respiratory Health

In the year of the study being reported here (Year II, Jan. - Dec. 1980), a total of 3439 children had both a completed parental interview and a pulmonary function test session. The response rate for interviews was greater than 95% in all years, and the response rate for testing was greater than 92% for Years II and III, and greater than 89% in Year I.

Details of the prevalence rates for respiratory symptoms and the values of pulmonary function will be reported elsewhere. In this report, for the purposes of associating with the effects of particle size we have selected two indicators of respiratory health (cough, and episodes of bronchitis), and two indicators of pulmonary function, both adjusted for body size.

In order to make the link between the aerosol data and the health variables, the initial design of our study attempted to divide the sample geographically into areas with known differences in pollution. From Figure 1 we can see that the city is divided by the escarpment into upper and lower sections, and in addition we divided the city into east and west areas relative to a principal street. The industrial

core (IC) is located in the north part of the east lower area. Thus the city was divided initially into five areas, and it is apparent from Figures 3, 4 and 5 that both particle size and concentration increase from west to east and from south to north across the city. Figure 6 shows the prevalence of three symptoms (cough, wheeze, cold to chest) as defined by the questionnaire, and as obtained from the interviews with the parents of each child. The figure shows a cluster of five bars for each symptom, each bar representing the prevalence in a different area of the city. It is apparent that in general, the prevalence of each symptom increases from the west upper area to the industrial core. Superficially, this would appear to show a substantial effect of air pollution; however, we also found that the prevalence of such other factors as parental smoking increased in the same way. This is apparent in Figure 7 where the likelihood of being in a low income bracket, the use of gas for cooking, and the prevalence of smoking in the home increased progressively from the west upper area to the industrial core. These factors have previously been implicated in a reduction of respiratory health of children even without an effect of pollution. We have previously reported on this problem and the extent to which it might have influenced our data, and for that reason found it necessary to use a method of analysis which

was more complex than the demonstration of simple independent associations (i.e. not adjusted for confounding factors) between health effects and air pollution (10).

Relative Strength of Environmental Effects

We chose to analyse these interdependent associations using stepwise multiple regression techniques. Initially we found a variety of independent variables that had shown at least one significant association with health-related outcomes. A list of these independent variables is given in Table I. We found, for example, that when examined as a single variable, gas cooking was associated with an increased prevalence of colds going to the chest. In the multiple regression analysis, all variables are introduced together and the computer selects the variables with the strongest and most significant effects, rejecting others that are highly correlated with the one selected. For continuous dependent variables, multiple linear regression was used; for binary dependent variables such as symptoms, the multiple logistic regression was used. A variable was said to be significant if the probability of a chance association was less than 0.05 (α=0.05).

Symptoms

The results of the logistic regression for symptoms are shown in Table II. The logistic regression calculates the influence of an independent variable on the probability of occurrence of an outcome variable according to the function shown in the Table. For example, two independent variables that were found to be significantly associated with the probability of a child having a cough during the day or night were: maternal cough, and hospitalization as an infant with respiratory disease. All other factors included were not significantly associated. In the table, the "p" value relates to the probability of the association being by chance (not the probability of the cough). Also in the table it can be seen that the probability of a child having an episode of cough and phlegm lasting at least three weeks (bronchitis) was significantly associated with three factors, maternal smoking being added to the other two.

Pulmonary Function

One of the measurements frequently made in respiratory epidemiological surveys is that of Peak Expiratory Flow (PF). Since the magnitude of PF is dependent on the size

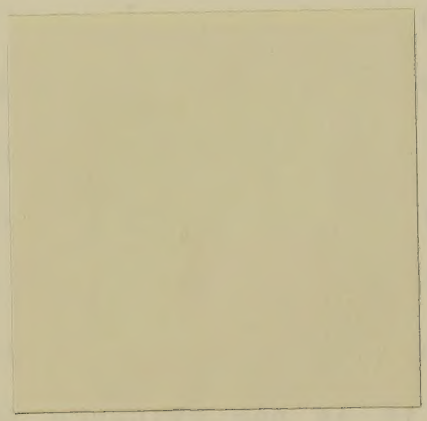
of the child, we have adjusted the values to the mean height of all the children (APF). In the example shown in Table III, the first equation shows that APF is reduced with an increased magnitude of the fine fraction. In addition, if the child shares a bedroom APF would be further reduced. Only these two variables were found to have an effect on adjusted peak flow. The MEF 75 is also a function of body size and must be suitably adjusted. From Table III, it may be seen that AMEF75 is significantly affected by three factors: maternal smoking; hospitalization of the child as an infant; and increased particle size. The observation with respect to particle size (MMD) should probably be interpreted in another way, however. It would be more logical to expect a reduction in function associated with particle exposure, and thus it could equally be said that MEF75 is reduced with a reduction in particle size, which would be consistent with the findings for peak flow.

DISCUSSION

We have carried out a study of the effects of ambient aerosol pollution and other environmental factors on the respiratory health of over 3500 school children in an industrial city. We have demonstrated a substantial

gradient in aerosol concentration and aerodynamic mass median diameter over the area in which the children live and attend school. By taking advantage of this gradient in exposure, and by assessing a variety of independent variables, we have shown that there are strong effects of the home environment, particularly of maternal smoking, and significant consequences of severe respiratory disease in infancy (which has been shown by others to be associated with smoking in the home) (11). We have also demonstrated effects of the fine fraction of aerosol pollution. Although this is based on a cross-sectional analysis of one year of data from the study, it is new evidence that the fine fraction of urban aerosol is associated with a degradation of pulmonary function in school children. There have been many studies of the effects of air pollution on the respiratory health of children (12-21), but only one has measured (but not yet reported) the effects of particle size (22).

Kotchmar and McMullen have reported in this symposium that for 5 U.S. cities which they studied, they could not find statistically significant differences amoung 3 sites where TSP and Fine Particulate were measured in a given city, for a 60 day sampling period (23). They concluded on this basis that one site would be suitable for monitoring in



these cities, and they suggest that the "cities represent a spectrum of types of particles and conditions leading to their emission, given some confidence to the greater application of this finding." Our observations for an industrial city with far from uniform terrain (lake on one side, 100 m escarpment parallel to the lake in the middle) indicate that the assumption of uniformity of particle size across the city is not valid in more than half of the cases in which we have data from more than 4 sites. It appears, however that spatial coefficients of the sites in our study were never significant in the months of July and August for the 3 years studied, and thus a statistically significant gradient could not be demonstrated for these months with the model we used. It is still possible that in these months a simpler model would show a site-related difference. This finding suggests that studies to determine the number of sites required for exposure estimation need to be done for each city for which exposure is desired, and should encompass at least 1 year to deal with seasonal differences in distribution.

In both the stepwise multiple logistic regression for symptoms and the stepwise multiple linear regression for pulmonary function, we observed that both hospitalization as an infant with respiratory disease and maternal smoking

were considered to be significant in three of the four relationships examined. We also observed that hospitalization for respiratory disease as an infant was also associated with maternal smoking. One might be led to suspect that only one or the other of these independent variables should be left in the relationship, but not both. However, the method used first takes into account the association between the two independent variables, and examines the remaining variability for its relationship to the dependent variable separately (24).

We have previously reported that indices of the Forced Vital Capacity (FVC) have greater discriminatory power in separating children with symptoms from those without, compared to tests using nitrogen washout (25). For the purposes of this report we have included the results of measurements of two pulmonary function variables (PF and MEF75) adjusted for height. We also examined other indices of the FVC, such as FEV_1/FVC, and height-adjusted FEV_1, MMEF, and MEF50. Three tests (FEV_1/FVC, MMEF, and MEF 50) all showed an effect of early hospitalization, and maternal smoking. Two showed effects of particles, which were just significant at the 0.05 level; MMEF showed an association with TSP, and FEV_1 an association with MMD. Both associations were positive, suggesting the interpretation

of a "fine particle effect", as we have evidence of a positive association between TSP and MMD. Thus the results of the other pulmonary function tests which we carried out are similar to those we presented in more detail, but the strength of the associations were less.

The same general points can be made about respiratory symptoms. The other symptoms that were examined, such as presence of any cough (not necessarily chronic), persistent wheeze, or colds "going to the chest" were all associated with hospitalization as an infant and maternal cough or maternal smoking. "Cold going to the chest" was associated with all 3 of the above, as well as concentration of TSP above the median (50 ug/m^3), but only at the 5% level. Symptoms were not associated with our measures of particle size.

Many studies (including some reported at this Symposium) have shown changes in pulmonary function associated with exposure to specific aerosols, such as sulphate, or sulphuric acid. None of these studies, for ethical reasons, have been carried out on children, who have a developing lung which may be more sensitive to damage by inhaled toxic material. Furthermore, the results of exposure have been observed either concurrent with or

immediately following exposure. Under these conditions it is possible to speculate on the physiologic interpretation of changes in pulmonary function produced acutely. The finding of a reduction in Peak or Maximum expiratory Flow associated with an increase in chronic exposure to the fine fraction of outdoor ambient aerosol in children with a mean age of 10 yr is much more difficult to interpret from a physiological point of view.

The physiological factors which influence flow during a maximum expiration are still not fully understood, but include the effort of expiration, elastic recoil of the lung, the upstream resistance (26), the viscoelastic properties of the airway wall and the density of the gas in the airway (27). When true flow limitation by dynamic compression of the airway has developed (usually after exhalation of the first 10 to 20% of the forced vital capacity), the interaction of these factors, although complex, becomes stable and predictable. This is much less so for the flow during the first 100 milliseconds of a maximum expiration, the period during which peak flow (or maximum expiratory flow) occurs. It is usually accepted that the major determinant of the maximum value obtained is the effort exerted by the subject. For this reason this test has not been well regarded for survey purposes,

although it is useful in a clinical context for 'following' specific patients to assess the effect of therapy.

Thus the physiological reason for the reduction in peak flow that we observe in children with a greater of aerosol is unclear, as it might be due to one or a combination of several factors which we have not separately addressed.

SUMMARY

We have operated a network of Andersen 2000 cascade impactors in the industrial city of Hamilton, Ontario, Canada, and have demonstrated statistically significant gradients in particle size over the city. We have carried out at the same time in the same city an epidemiological study of respiratory health of over 3500 elementary school children in whom we have measured pulmonary function and the prevalence of symptoms, as well as characteristics of the home environment, including family smoking. We have developed a method of determining exposure of each child to particles which depends on the location of the child and the characteristics of the gradient we have observed. We found strong negative health effects of the home environment, particularly maternal smoking, and relatively weak effects of the ambient air quality. We did observe,

however that there was a statistically significant association between decrement in pulmonary function and an exposure to fine particles.

CONCLUSION

There is a good theoretical basis for the hypothesis that particle size and chemical composition are important determinants of particle toxicity (28). There has been much recent discussion in the U.S. (29) which has led to the promulgation of a new standard for suspended particulate material based on a size criterion by the EPA (30). Although work reported at this Symposium suggests that acute effects of sulphate aerosols at common ambient concentrations are unlikely to be observed (31), the results of the extensive exposure work by Kagawa (32) seem to point to the development of ultrafine aerosols associated with SO_2 exposure. It may be that it is chronic exposure to the very fine fraction of sulphate aerosol which is associated with a decrement of respiratory function, such as that described by Lioy and Lippman (33) and the increased rate of admission to Ontario hospitals for acute respiratory disease reported by Bates and Sizto (34) at this symposium. The results of this study support the idea that the "respirable" (<2.5 um) rather than the

"inhalable" (<10 um) fraction of urban aerosol is the more important, when considering impact on the respiratory health of children.

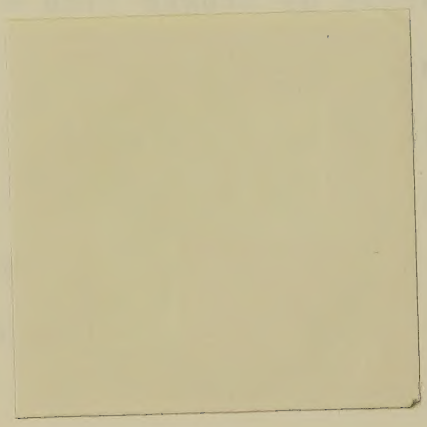
LEGENDS FOR FIGURES

Figure 1 (A) Outline map of Hamilton, Ontario. City limits shown as broken line with two dashes; study area boundaries by broken line with dots. Grid lines are Universal Transverse Mercator at 5000 m intervals. Grid reference for the intersection point at "WL" is 590000 E, 4790000 N (Zone 17T). Crosses are TSP sites; open figures are PSD sites; a number of PSD sites are located at TSP sites.

(B) Same area as (A), but showing locations of schools ("S") as well as sampling sites

Figure 2 Relationship of concentration of fine fraction obtained from Sierra 245 dichotomous sampler (DICHOT) to fine fraction obtained from Andersen cascade impactor (AND 2000) at Barton/Wentworth site in Hamilton. Data from Oct. 1983 to Sept. 1984 inclusive (6-day cycle).

Figure 3 Distribution of particle size over the City of Hamilton for May, 1980. Contours of MMD



derived from the response surface. Mass median diameter in micrometers may be obtained from the small numbers defining the isopleth by dividing by 2.

Figure 4 Distribution of particle size over Hamilton: averages for three years. Contours derived from the response surface for yearly (12-month arithmetic mean) of MMD; isopleth system as in Figure 2.

Figure 5 Distribution of fine fraction: FF, (<3.3 um) and coarse fraction: CF, (≥3.3 um) of particles over Hamilton for 3 years. Concentration defined by the isopleths in ug/m^3 are obtained by multiplying isopleth number by 10.

Figure 6 Differences in prevalence of 3 respiratory symptoms in 5 areas of Hamilton found to be significant by X^2 test ($p<0.05$). Areas shown by different hatching. See Figure 1A for location of areas.

Figure 7 Differences in prevalence of environmental covariables in 5 areas of Hamilton found to be significant by X^2 test ($p<0.05$). Areas shown by different hatching. See Figure 1A for location of areas.

Table I. Independent Variables in Regression

- Hospitalization at < 2 Yr. of age for Respiratory Illness
- Mother Has Cough
- Mother Smokes / # of Cigs/Day
- Father Smokes / # of Cigs/Day
- Father Has Cough
- Family Income
- Father's Occupation
- Mother's Occupation
- Gas Cooking
- Child Shares Bedroom
- Family: # Cigs/Day
- Total Suspended Particulates
- Mass Median Diameter
- Coarse Fraction
- Fine Fraction

Table II. Respiratory Health Logistic Regression

Function: P = 1 / (1 + e * * -[A + B1X1 + B2X2 + ...])

SYMPTOM	COEFF	COEFF VALUE	VARIABLE	SIGNIF OF COEFF	CODE Pres , Abs
Cough Day Or Night	A	-1.80	CONSTANT	(p<0.00001)	--- ---
	B1	0.44	MACOF	(p<0.00001)	1 , 0
	B2	-0.34	HOSP<2yr	(p<0.001)	1 , 2
Episode of Cough And Phlegm >3 Weeks	A	-1.69	CONSTANT	(p<0.00001)	--- , ---
	B1	0.35	MACOF	(p<0.00001)	1 , 0
	B2	-0.29	HOSP<2yr	(p<0.001)	1 , 2
	B3	0.19	MASMOKE	(p<0.003)	1 , 0

Table III: Pulmonary Function Stepwise Multiple Linear Regression

Function: PULM. FUNC. = A + B1X1 + B2X2 + ...

PULMONARY FUNCTION VARIABLE	COEFF	COEFF VALUE	VARIABLE	SIGNIF OF COEFF	UNITS OR CODE Pres , Abs
Height-Adjusted Peak Flow	A	4.220	CONSTANT	(p<0.00001)	--- ---
	B1	-0.010	FF	(p<0.00001)	ug/m^3
	B2	-0.049	SHAROOM	(p<0.04)	1 , 0
Height-Adjusted MEF 75	A	0.544	CONSTANT	(p<0.0001)	--- ---
	B1	0.125	MMD	(p=0.0001)	um
	B2	-0.002	MACIG	(p=0.0001)	Cigs/Day
	B3	0.067	HOSP<2yr	(p<0.02)	1 , 2

ACKNOWLEDGEMENTS

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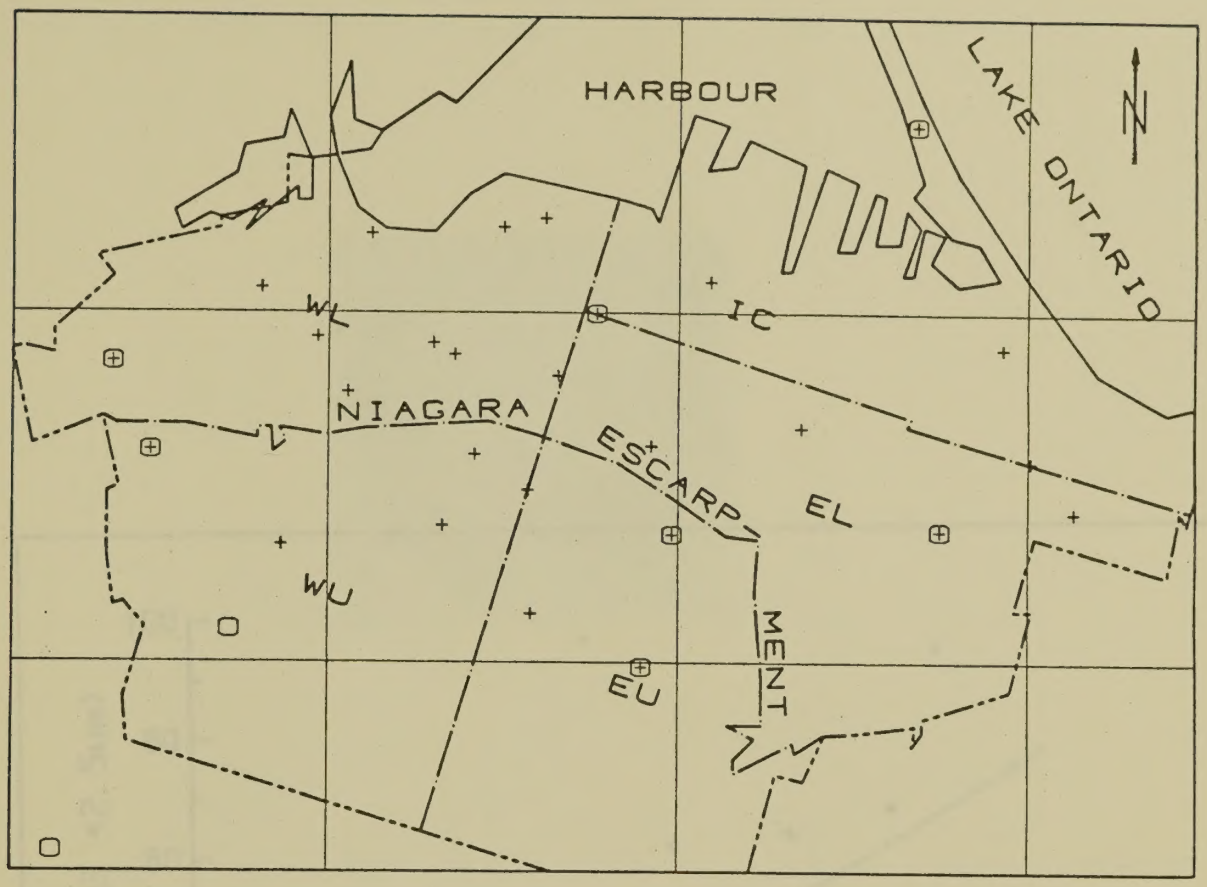

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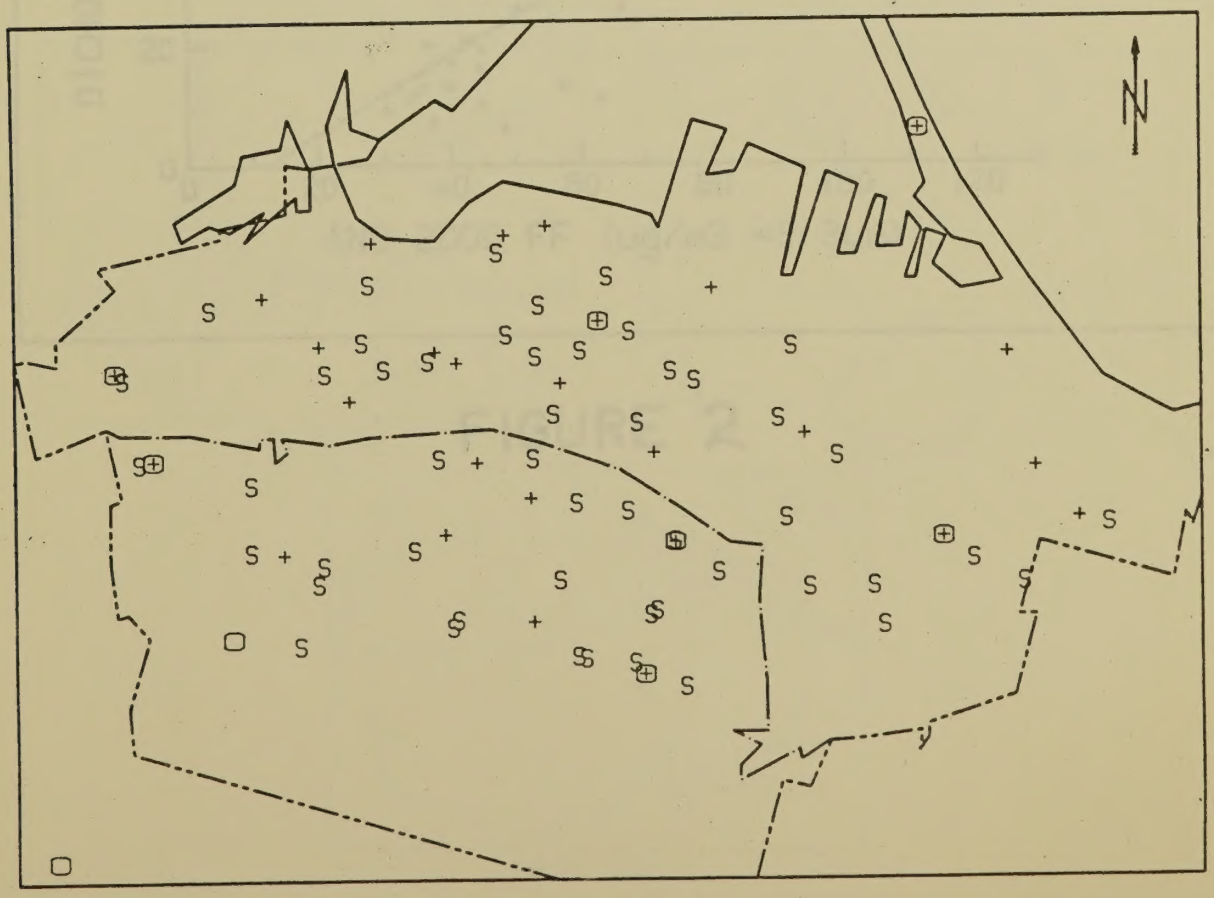

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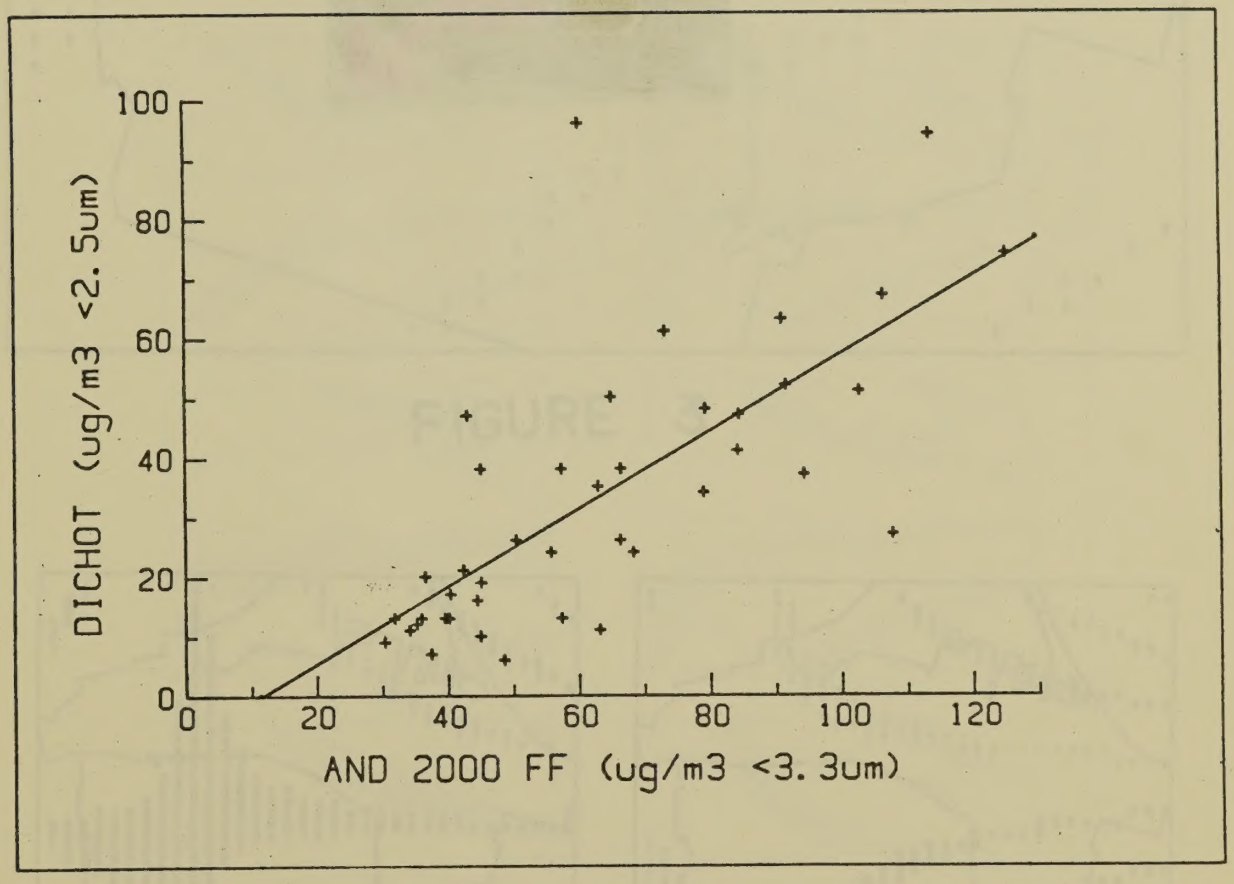
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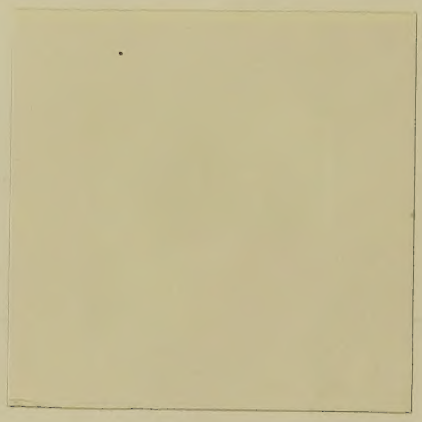
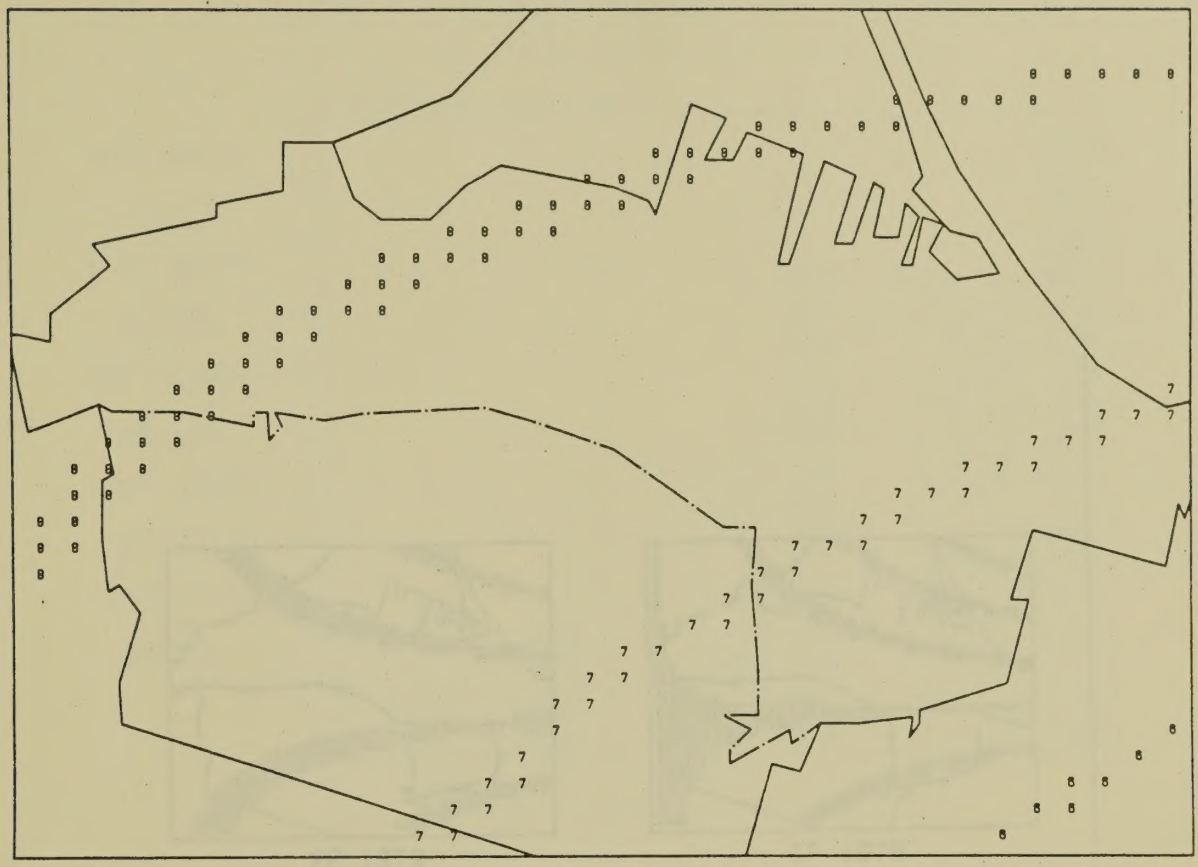
FIGURE 2





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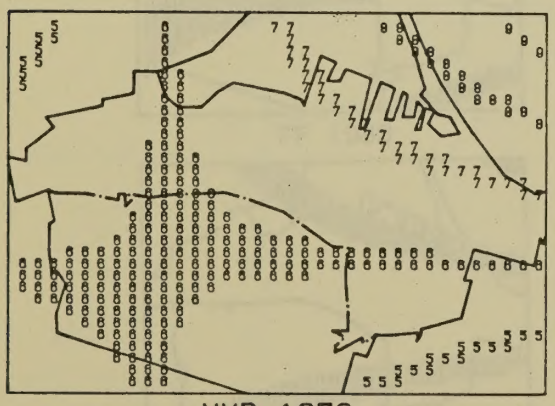

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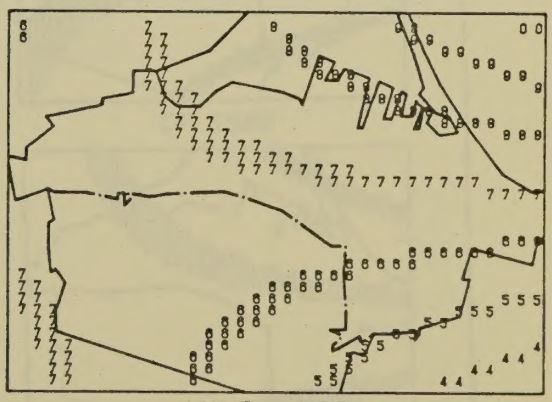

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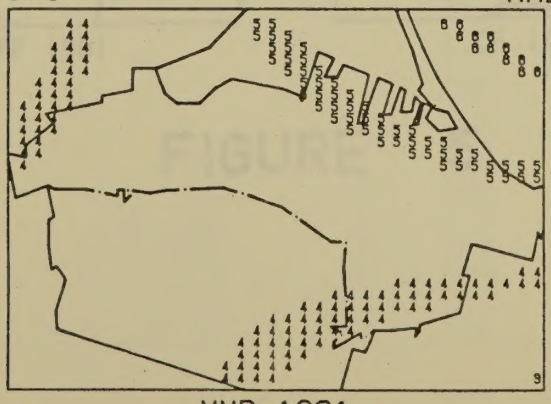

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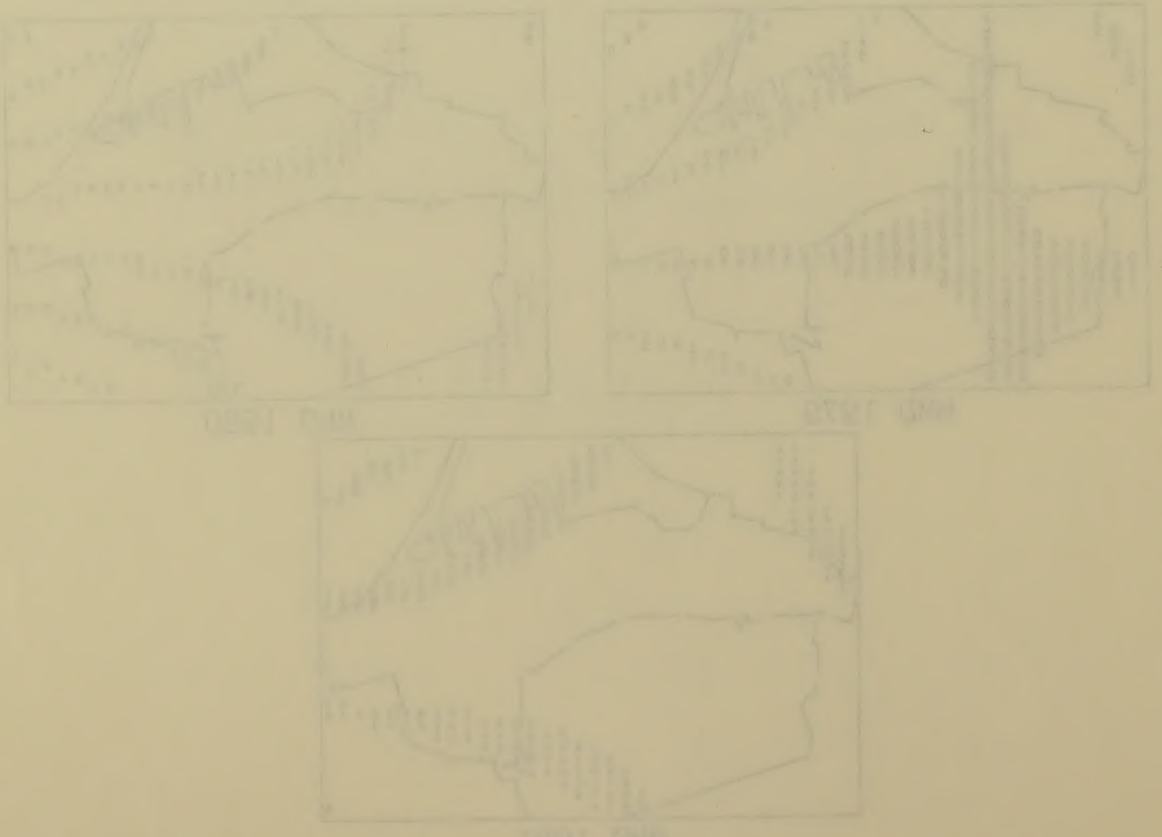

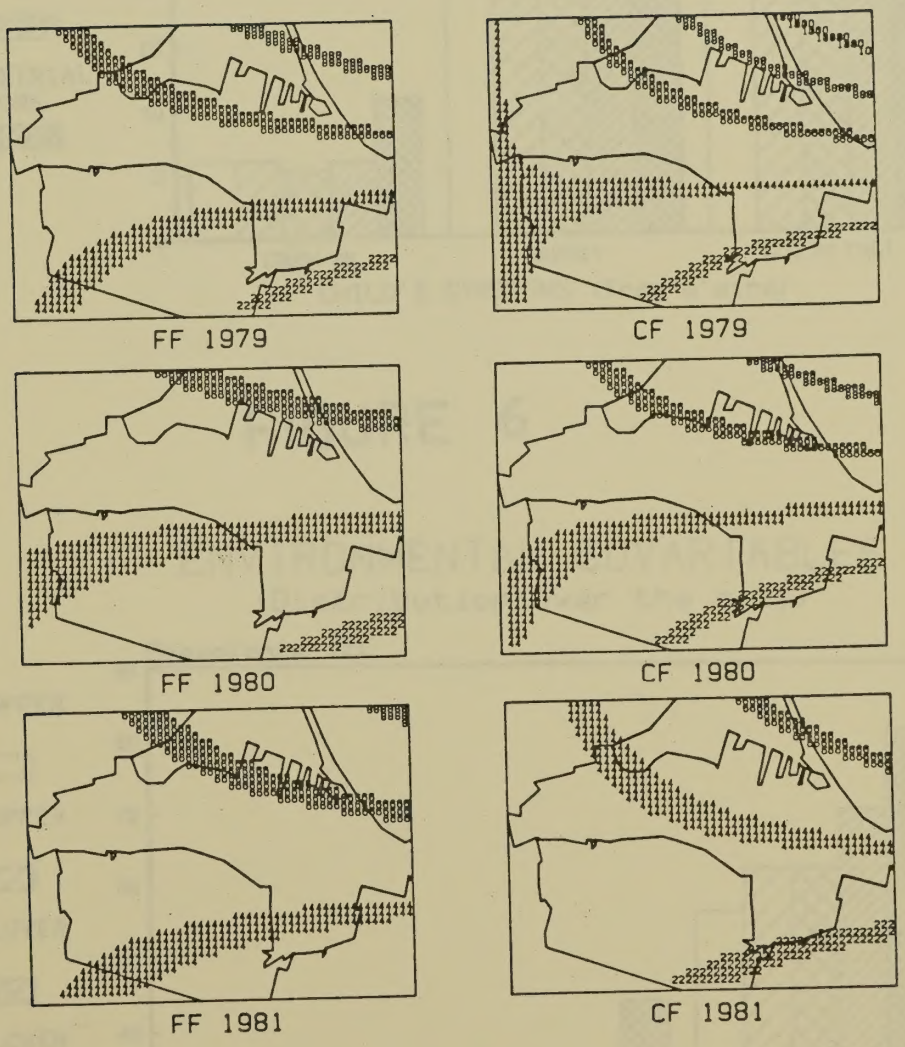

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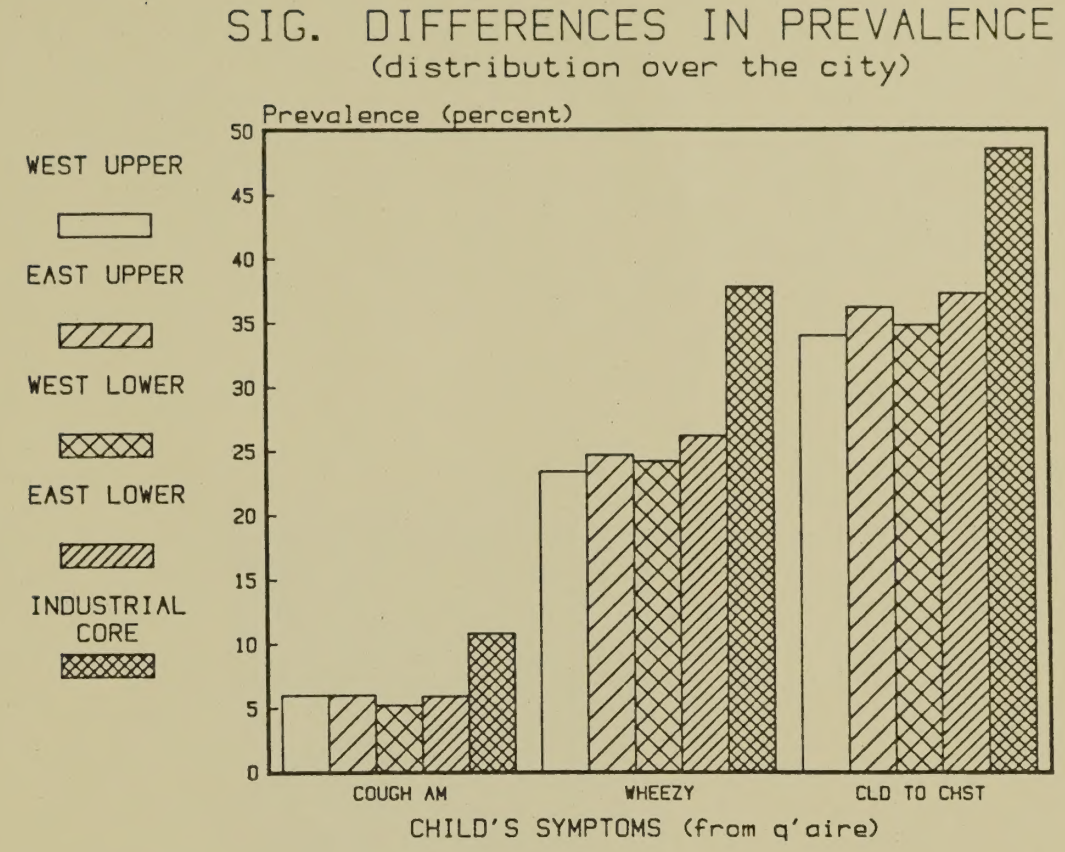

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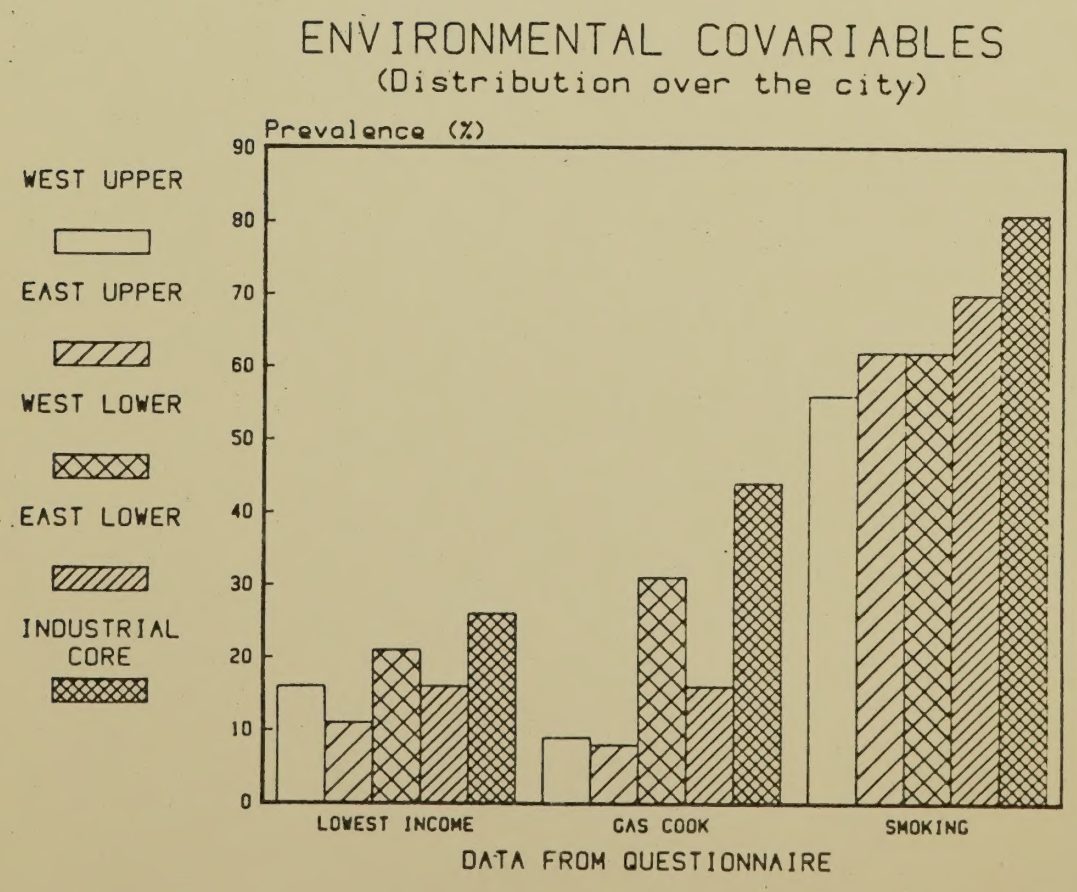

FIGURE 7

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